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SERUM TESTOSTERONE LEVELS AND ITS IMPACT ON DIABETIC PATIENTS

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ABSTRACT

The testosterone therapy in male patients has become clinical interest now days. Although there are a number of potential benefits of male testosterone replacement therapy, but yet there have not been any controlled trials of this therapy for type-2 diabetes. In present study we investigate the effect of testosterone replacement therapy by discussing a limited number of trials initially. The study was carried out with 70 hypogonadic patients at the age of 40 to 60 years selected from Life hospital situated in Guntur. Patients with the serum levels <200ng/dl were given 80mg Andriol orally and to patients with testosterone >200ng/dl were given 40mg/day was administered orally for 6 months to the male patients. The biochemical test and level of hormonal in blood was tested before the treatment, after three months and six months of the treatment. Initially the average testosterone level (204 ng/dl) were increased approximately two times (452 ng/dl) after six months of treatment. Hence, we found that testosterone therapy fasting blood glucose, postprandial glucose and glycosylated hemoglobin (HB1AC) reduced significantly. Therefore, the testosterone replacement therapy might have the beneficial effects on treating type-2 diabetes.

KEY WORDS: Testosterone, Diabetes, Hypogonadism.

INTRODUCTION:

The testosterone hormone plays the key role in masculine growth and development during puberty. However, testosterone level in blood was started decline probably at the age of 30 years irrespective with the health condition (Gary et.al 1994). The inefficient production in male body leads to hypogonadism (Vermeulen, 2001) that can be causes for loss of fat free mass (FFM), skeletal muscle growth, and gain in fat mass (FM). The correlations between these aspects of ageing and the known role of exogenous testosterone, it was hypothesized that the testosterone therapy in ageing men will result in favorable changes in body composition and may improve metabolic status by reversing the loss of FFM and gain in FM (Bhasin et al., 1996, 1997)

Testosterone levels are found to be less in type-2 diabetic, obese and patients with insulin resistant syndrome (Marin et al., 1992; Boyanov et al., 2003). There is further evidence that low testosterone levels are the risk factor of the later development of metabolic syndrome and diabetes (Grossman et al., 2008). It was suggested that administration of testosterone in hypogonadic person can improve the glucose tolerance.

The present study was designed to understand the changes in clinical conditions of diabetes, and hypogonadism after the testosterone replacement. An attempt was made to study the changes in clinical conditions of diabetes, obesity and hypogonadism before and after the testosterone replacement. However, limited trials of studies were conducted in subjects who have low testosterone levels associated with diabetes of age range of 40-60. The main objectives of the study were made to know about the clinical status of the hypogonadic subjects of 40-50 and 50-60 age groups before and after testosterone administration. Furthermore, the clinical status of diabetes before and after testosterone treatment in 40-50 and 50-60 age groups were studied.

MATERIALS AND METHODS:

Patients

The study was confined in Guntur district and its' surrounding areas. In the present study, 100 male patients between the age of 40-60 years were screened for diabetes. Out of them, seventy patients diagnosed with type-2 diabetic, and hypogonadism were chosen for the study.

Testosterone readily available in the market by the trade name of Andriol testocaps (Organon company Ltd., Mumbai) was selected in the study. Each testocapsule contains testosterone undecanoate 40 mg. Andriol 40-80 mg was administered orally for six months to the male patients after a basal anthropometric, biochemical, and hormonal evaluation. Patients with serum testosterone levels<200ng/dl were given 80mg andriol orally and patients with serum testosterone >200ng/dl were given 40mg per day as per practitioners prescription. The response therapy was monitored by clinical examinations after three months and 6 months of testosterone treatment, fasting blood glucose (FBG), post prandial blood glucose (PPG), glycosylated hemoglobin (Hb1Ac), triacylglycerol (TGL), high density lipoprotein cholesterol (HDL), total cholesterol, and testosterone evaluation were done. During the above course patients were advised to take low calorie diet and to do moderate physical activity.

All biochemical and hormonal analyses were performed in fasting blood samples of all patients. After an overnight fast, a fasting blood sample was obtained between 8 and 10 am. Blood samples were collected in ethylene diamine tetra acetic acid-coated venipuncture tubes and maintained at 2-80c until assayed and the plasma was separated by centrifugation at 3000 rpm for 15min.

The whole data obtained was divided into 3 samples, the first sample categories as patients and their parameters before treatment, second samples was categorized from the patients those who have received the dose for 3 months with restricted diets and sample third are categorized from the patients and their parameters after receiving the dose for 6 months with restricted diet.

Based on the age of patients were divided into two groups. Group A comprises of 36 patients at the age of 40-50 years old and Group B consists of 34 patients at the 50-60 years old. Effect of testosterone on diabetes was studied by estimating fasting blood glucose, post prandial blood glucose, Hb1Ac by glucose oxidase peroxidase method and also by estimating total cholesterol by cholesterol oxidase peroxidase method, HDL cholesterol by phosphotungstate method, TGL by glycerol 3phosphate oxidase peroxidase method and measuring anthropometric parameters like weight, height and BMI. Testosterone was estimated by immunoassay. The results were validated through statistical analysis.

RESULTS:

Seventy hypogonadic diabetic patients were selected to study the effect of testosterone on diabetes. Testosterone in the form of testocaps was given for 6 months. Blood samples were collected 3 times, before treatment, after 3 months and after 6 months of treatment and the biochemical, hormonal and anthropometric measurements were also analyzed.

Average Testosterone values were 204 ng/dl initially and after testosterone treatment for about 6 months the values increased to 452 ng/dl. Both the age groups responded equally on testosterone administration (Table No. 1). When percentage increase was calculated group wise the rates of increase of testosterone on andriol administration were uniform for both 40-50 and 50-60 years old age groups.

Serum testosterone levels are 17% lower in men with clinical depression than who did not have depression. In this study we observed that testosterone levels before treatment with testosterone positively correlated with sociability and a sense of wellbeing. After therapy was initiated, positive mood increased and all measures of negative mood declined.

Results revealed the action of testosterone on fasting blood glucose and post prandial glucose. Fasting blood glucose decreased by 12% in 40-50 age group and 8% in 50-60 age group in first 3 months. In last 3 months the percentage reduction was 20 percent in both age groups which show that on testosterone dosage glucose tolerance was maintained (Table No.2). The average post prandial glucose was 229.7mg/l in sample 1 and it reduced to 189.5mg/dl and 150mg/dl in sample 2 and sample 3 respectively (Table No. 3). When percentage reduction was calculated age wise it was observed that the reduction was more in 40-50 age group than in 50-60 age group. The Hb1Ac percentage reduction was more in 40-

50 age group than in 50-60 age group at initial three months and also at last 3 months (Table No.4).

The effect of testosterone on lipids was found to be significant.. There was a significant decrease in total cholesterol and TGL and a small but significant rise in HDL after successive testosterone treatment. When the total cholesterol percentage decrease was calculated age wise, the percentage reduction was less in 40-50 years age group compared to 50-60 years age groups and When percentage increase was calculated for HDL cholesterol it was more in 40-50 years age group than in 50-60 years age group. At the age group of 40-50 yearsTGL level decreased more in first 3 months where as in 50-60 years age group TGL was decrease gradual during entire 6 months period.

Results show that glucose tolerance improved in age group of 40-50 than in 50-60 year age group in response to testosterone administration and lipid profile and anthropometric measurements responded well in 50-60 age group than in 40-50 age group. The group wise waist measurement indicated that percentage reduction in weight was less in group A compared to B. However the of reduction in hip value was negligible during 6months of course. When waist to hip ratio was analysed it was observed that it reduced more in age group of 40-50 than in 50-60 age groups.

When statistical regression technique(Table-5) was applied by considering PPG as the dependent variable and the rest of all parameters as independent variables at 5% level of significance, it was observed that in sample-I there is highly significant influence of all the predictors (independent variables) on dependent variable(Table No.5). Similarly the same study was extended for sample-II and was also found that the dependent variable- PPG was influenced very significantly by all the independent variables. For sample-III also the regression value was highly significant ($p< 0.01$). (Table No.6). To study the nature of influence intrinsically stepwise regression was applied on the data. The dependent variable PPG was much influenced by Hb1Ac, weight, TGL, and testosterone in sample-I very significantly. But, in sample-II only Hb1Ac and weight showed much influence on PPG as its 'R'-Square value was high (r2 -.891). In sample-III the variables Hb1Ac, height and HDL cholesterol showed their influence on PPG significantly.

Considering the level of testosterone as the dependent variable and rest of all parameters as the independent variables it was observed that in all three groups the influence of the considered independent variables is not significant on the levels, for $p<0.05$, of testosterone(Table No.7,8). The stepwise regression also the influence of different combinations of independent variables is not significant.

Negative correlations of all the three groups were observed by evaluating the coefficients of correlations among testosterone, FBG, PPG, for sample-I. The less testosterone level switching the more PPG and FBG concentration in sample-I and vice versa in sample-III was observed. In sample-I, it was observed that diabetic men over the age of 40 had low serum testosterone with high FBG, PPG and Hb1Ac. There was a strong correlation between low free testosterone level and diabetes. Low total testosterone level was more strongly associated with elevated BMI and elevated waist-to-hip ratio than with diabetes. In this study we observed that men with age more than 40 years would likely to have low testosterone levels with diabetes. In this study it was observed that individuals with BMI>30 had elevated Hb1Ac and serum testosterone below normal range in sample-I. On successive treatment serum testosterone rose to normal range with a decrease in Hb1Ac and BMI. This might be due to testosterone as it inhibits lipoprotein lipase activity resulting in reduced triglyceride uptake by adipocytes and decreased adiposity.

DISCUSSION:

The goal of testosterone therapy is to achieve and maintain ideal serum testosterone levels. The effect of exogenous androgens on the behavior of eugonadal men have been studied in several investigations (Anderson, 1994). Studies with varying doses of testosterone have found that while (dosage >500mg) may increase manic tendencies, aggression, sexual interest, and euphoria,and (dosage of 300mg may produce twice the physiological concentrations resulted the increase sexual interest with no increase in aggression (Anderson, 1994).

In a study, an age related decrease in total and bioavailable testosterone, as well as dihydrotestosterone, showed an increase in measures of depression which might be due to the correlation with the testosterone level (Barrett-Connor, 1990). Testosterone doses administered to type 2 diabetic patients for the period of 6 months induced changes in fasting blood glucose, postprandial glucose and Hb1Ac. The above observations illustrate the effect of testosterone on diabetes. Testosterone is the standard therapy for male patients with hypogonadism (Nieschlag, 2006).

Low total testosterone or SHBG levels are associated with type 2 diabetes, independent of age, race, obesity, and criteria for diagnosis of diabetes (Corona , 2011). In longitudinal studies, low serum total, SHBG and free testosterone were independent predictors of type 2 diabetes (Corona, 2011; Vikan 2010). Studies by Chaoyang et al., 2010 also demonstrated that SHBG and testosterone were strongly associated with metabolic syndrome and insulin resistance in men.

A low serum testosterone concentration is associated with insulin resistance syndrome (Svartberg et al., 2004, Traish et al.,2009). In this study shows that low testosterone is associated with elevated levels of FBG, BMI. As testosterone levels are inversely related to blood glucose levels we tried to study the reversal of diabetic condition by administration of oral testosterone.

Abdominal obesity is a significant determinant of insulin sensitivity and is likely to impair hepatic and peripheral insulin sensitivity in obese patients. Increased adipose tissue is associated with an increase in the enzyme aromatase that converts testosterone to estradiol. Schneider et al. reported that Estradiol levels are two-fold higher in obese men (1979). Dexamethasone has been found to suppress estradiol and estrogen levels suggesting that higher estrogen levels in obese men may be dependent upon adrenal hormones (Zumoff, 1988).

The studies demonstrated that men with diabetes are significantly more likely to have hypogonadism (Zitzman et al., 2006, Kapoor et al., 2005). Testosterone levels are inversely related to higher hemoglobin A1c levels and hemoglobin A1c levels were significantly higher in men in the lowest quintile of testosterone levels (Svartbeg et al., 2004; 2007; Stanworth and Jones, 2009). This data is correlated with our present finding.

SHBG levels also inversely correlate with obesity in cross sectional studies (Stefanik et al., 1987; Haffner et al., 1993; Osuna et al., 2006). In general, SHBG concentrations increase with age. Thus the rate of decline of total testosterone was greater for overweight men than for thin men due to the lower rate of increase in SHBG (Mohr et al., 2006).

Singh et al., (2003) provides information about the mechanisms of androgens on changing body compositions in animal model The report suggested that androgens caused differentiation of mesenchymal pluripotent cells through the myogenic pathway and inhibited the progression of the adipogenic pathway. This mechanism was accepted as an explanation for the changes observed in muscle and fat mass during testosterone supplementation. Testosterone exerts its effects by means of hormones originating from adipose tissue through unexplained mechanisms. The existence of a close relationship between sex steroids and adipose tissue can be demonstrated by these observations.

Heufelder et al., 2009 showed the beneficial effects of supervised diet and exercise treatment on glycemic control, components of metabolic syndrome and newly diagnosed type 2diabetes. These effects were even greater when transdermal patches of testosterone was added, which resulted in 80%of the patients showing conversion from the metabolic syndrome, reaching all set targets of glycemic control. This study shows that glucose tolerance improved in age group of 40-50 than in 50-60 year age group in response to testosterone administration and lipid profile and anthropometric measurements responded well in 50-60 years age group than in 40-50 years age group.

In a recent study, Jones et al., (2011) demonstrates that Transdermal testosterone replacement therapy in hypogonadal men with type 2diabetes and/or metabolic syndrome improves insulin resistance several cardiovascular risk factors mostly lipoprotein a(Lpa), total cholesterol and LDL cholesterol. The study also suggests that Lpa is the most atherogenic component of the lipid profile and testosterone is the only therapy that is known to reduce levels. All these findings strongly support our observations.

CONCLUSION:

The use of testosterone in therapy has become interest of both the medical and lay communities. Present study illustrates the challenging in diagnosing and treating type-2 diabetes, insulin resistance syndrome and obesity in the setting of hypogonadism. Given the unique physiological connections between adiposity, insulin resistance, obesity and hypogonadism i.e. low testosterone levels, suggests that testosterone replacement therapy can improve the situation and further reduces the risk related to cardiovascular diseases.

In conclusion, hypogonadism in men may lead to the development of the insulin resistance syndrome or diabetes through various mechanisms including changes in body composition; androgen receptor polymorphisms; glucose transport; and reduced antioxidant effect. It is suggested that the increased risk of diabetes and insulin resistance syndrome seen among hypogonadal men is factorial and testosterone replacement may mediate the effects at multiple levels.

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Table No. 1 Testosterone percentage increase group wise

GROUPS	PERIOD	PERCENTAGE INCREASE
A	First 3 months	14%
	Last 3 months	14.6%
B	First 3 months	14%
	Last 3 months	14%

Table No. 2 FBG percentage reduction group wise

GROUP	PERIOD	PERCENTAGE REDUCTION
A	1st 3 months	12% reduction
	Last 3 months	20% reduction
B	1st 3 months	8% reduction
	Last 3 months	20% reduction

Table No. 3 PPG percentage reduction group wise

GROUP	PERIOD	PERCENTAGE REDUCTION
A	1st 3 months	17% reduction
	Last 3 months	21.3% reduction
B	1st 3 months	16% reduction
	Last 3 months	19.9% reduction

Table No. 4 HB1AC percentage reduction group wise		
GROUP	**PERIOD**	**PERCENTAGE REDUCTION**
A	1st 3 months	13.1% reduction
	Last 3 months	16.1% reduction
B	1st 3 months	12.2% reduction
	Last 3 months	14% reduction

Table No. 5 REGRESSION OF PPG SAMPLE-I				
Model	**R**	**R Square**	**Adjusted R Square**	**Std. Error of the Estimate**
1	.974[a]	.949	.937	6.651

a. Predictors: (Constant), FBG, HDL, W/H,HEIGHT, AGE, HBIAC,TESTOSTERONE, CHOLESTEROL, TGL, WAIST, BMI, WEIGHT, HIP

TABLE No. 6 REGRESSION OF PPG SAMPLE-III				
Model	**R**	**R Square**	**Adjusted R Square**	**Std. Error of the Estimate**
1	.883[a]	.780	.729	7.773

a. Predictors: (Constant), FBG, HDL, W/H,HEIGHT,AGE, HBIAC, TESTOSTERONE, CHOLESTEROL, TGL, WAIST, BMI, WEIGHT, HIP

TABLE No.7 REGRESSION OF TESTOSTERONE SAMPLE-I				
Model	**R**	**R Square**	**Adjusted R Square**	**Std. Error of the Estimate**
1	.665[a]	.442	.312	34.394

a. Predictors: (Constant), CHOLESTEROL, WAIST, AGE, PPG, WEIGHT, FBG, HDL, W/H, TGL, BMI, WEIGHT, HBIAC, HIP

TABLE No. 8 REGRESSION OF TESTOSTERONE SAMPLE-III				
Model	**R**	**R Square**	**Adjusted R Square**	**Std. Error of the Estimate**
1	.599[a]	.358	.210	28.452

a. Predictors: (Constant), CHOLESTEROL, WAIST, AGE, PPG, HEIGHT, FBG, HDL, W/H, TGL, BMI, WEIGHT, HBIAC, HIP

TABLE No.9 ANOVA

Table No.4.48 Anova: Two-Factor Without Replication				
SUMMARY	**Count**	**Sum**	**Average**	**Variance**
Sample1 Average	13	1466.858	112.8352	8622.009
Sample 2 Average	13	1467.123	112.8556	10281.65
Sample 3 Average	13	1452.565	111.7357	14895.6
WEIGHT	3	257.1	85.7	46.19816
HEIGHT	3	198.2571	66.08571	0
BMI	3	91.91429	30.6381	6.19619
WAIST	3	228.1429	76.04762	105.309
HIP	3	215.8	71.93333	0.291905
W/H	3	3.207714	1.069238	0.020847
FBG	3	363.4857	121.1619	478.7421
PPG	3	569.3286	189.7762	1586.916
HBIAC	3	22.58	7.526667	1.294748
TGL	3	535.8571	178.619	741.2707
HDL	3	114.7571	38.25238	7.170272
CHOLESTEROL	3	797.2857	265.7619	578.8444
TESTOSTERONE	3	988.8286	329.6095	15436.62

TABLE No.10 MEAN PROCEDURE–SAMPLE-I

Variable	Label	N	Mean	Std Dev	Minimum	Maximum
AGE	AGE	70	52.0857143	5.5370762	40.0000000	60.0000000
WEIGHT	WEIGHT	70	92.6285714	11.2574245	76.0000000	122.0000000
HEIGHT	HEIGHT	70	66.0857143	4.8476585	58.0000000	76.0000000
BMI	BMI	70	33.2000000	2.9713609	26.0000000	39.0000000
WAIST	WAIST	70	85.9285714	10.9442877	60.0000000	120.0000000
HIP	HIP	70	72.4285714	11.8181529	50.0000000	100.0000000
W/H	W/H	70	1.2104286	0.2183830	0.9200000	1.8000000
FBG	FBG	70	140.7142857	24.0163850	94.0000000	220.0000000
PPG	PPG	70	229.7142857	26.5062983	160.0000000	310.0000000
HBIAC	HBIAC	70	8.6614286	0.7423607	6.5000000	11.0000000
TGL	TGL	70	206.3000000	27.0888768	150.0000000	280.0000000
HDL	HDL	70	35.7428571	5.0351970	25.0000000	47.0000000
CHOLESTEROL	CHOLESTEROL	70	290.0000000	24.0518763	250.0000000	390.0000000
TESTOSTERONE	TESTOSTERONE	70	204.2428571	41.4642978	120.0000000	300.0000000

Table No. 11 The MEANS Procedure Sample-3

Variable	Label	N	Mean	Std Dev	Minimum	Maximum
AGE	AGE	70	55.5571429	8.2998790	40.0000000	70.0000000
WEIGHT	WEIGHT	70	79.0428571	10.3173355	62.0000000	112.0000000
HEIGHT	HEIGHT	70	67.4142857	6.5308473	58.0000000	89.0000000
BMI	BMI	70	28.2285714	2.7513972	22.0000000	34.0000000
WAIST	WAIST	70	65.4428571	10.5825064	50.0000000	90.0000000
HIP	HIP	70	71.3571429	11.0270231	50.0000000	90.0000000
W/H	W/H	70	0.9218571	0.1459233	0.7700000	1.7000000
FBG	FBG	70	97.5285714	10.3555928	40.0000000	120.0000000
PPG	PPG	70	150.0428571	14.9263270	99.0000000	180.0000000
HBIAC	HBIAC	70	6.3857143	0.3448527	6.0000000	7.2000000
TGL	TGL	70	151.8714286	14.7283828	110.0000000	185.0000000
HDL	HDL	70	41.0714286	2.7043788	35.0000000	49.0000000
CHOLESTEROL	CHOLESTEROL	70	241.8857143	11.7701943	216.0000000	270.0000000
TESTOSTERONE	TESTOSTERONE	70	452.7000000	32.0006114	390.0000000	550.0000000